CHANGES OF THE CELL BODIES IN THE FACIAL NUCLEUS AFTER FACIAL NERVE BLOCK

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SUMMARY
Facial nerve blocks were produced in four monkeys by insertion of a needle into the facial nerve trunk. The sequential changes of nerve cells in the facial nucleus were investigated 4 days, and 2 and 7 months later. Although the facial paralysis recovered in 2 months, considerable changes of cell bodies in the facial nucleus were still obvious which recovered completely only after 7 months. It is postulated that an optimum discharge is generated by those nerve cells in the facial nucleus during the recovery period which in clinical practice could result in 6 months of clinical remission from facial spasm.

Physical nerve-block (insertion of a needle directly to the bony facial canal to damage the nerve) developed by Wakasugi (1972a, b) has been regarded as the most effective treatment for facial spasm. Using this method we have treated 225 patients with hemifacial spasm in the Pain Clinic of Kyushu University Hospital. When the nerve block was applied, the spasm disappeared immediately. The facial paralysis which developed then recovered gradually in 1.5-2 months (fig. 1). Thereafter, the patients were symptom-free for about 6 months without any paralysis or spasm. However, relapse occurred in many cases within 8-10 months after treatment (fig. 2). Stimulated by these clinical observations, we observed the changes in the nerve cells of the facial nucleus following nerve block in Japanese monkeys.

METHODS AND HISTOLOGICAL PROCEDURES
Four male Japanese monkeys (4-10 yr old) were studied. Three of them were anaesthetized with ketamine hydrochloride, and a 22-gauge needle was inserted into the neural canal of the facial nerve to produce a nerve block (fig. 3). The needle was
FIG. 3. Facial nerve block in a Japanese monkey. The needle is inserted about 5 mm below the tip of the stylo-mastoid process, and moved parallel to the frontal plane at 30° to the sagittal plane to reach the stylomastoid foramen. The peripheral facial nerve becomes paralysed when the needle (arrow) enters the neural canal. The animal is unable to close its eye and the cornea is exposed by about 3 mm; a prominent angular paralysis is evident.

removed 30 min later after producing complete paralysis. One monkey was used as control. The animals were deeply anaesthetized and the tissues fixed by intracardiac perfusion with 10% formalin 5 litre at 4 days, and at 2 and 7 months after the nerve block. The brain was removed immediately, fixed in 99% alcohol and immersed in celloidin. The brain stem was cut serially in 30-μm sections in the transverse plane and stained with toluidine blue.

RESULTS

Normal characteristics of the facial nucleus in a 6-yr-old monkey weighing 12 kg

The facial nucleus was found as a group of neurons symmetrically located on the ventrolateral side of the abducens nucleus at the lower edge of the pons. This group of neurons appeared nearly circular in cross-section, and could be identified distinctly from the surrounding tissues. The nerve cells were stellate, spindly, or oval, with a single nucleus located mostly in the centre of the cell body. The nuclear membrane was prominent and the Nissl’s granules were lined along the perimeter of the cells with a thick concentration around the outer edge of the nuclei (fig. 4: 1).

Experimental monkeys

(1) A 5-yr-old monkey weighing 11.6 kg. Sacrificed 4 days after nerve block. The facial nerve of the blocked side was completely paralysed. The animal was unable to close its eye, with the cornea exposed by about 3 mm, and was unable to drink water from the water bottle because of angular paralysis. The nerve cells of the facial nucleus were circular with the nuclei shifted to one side of the cells. Dispersion of the Nissl substance, with decrease in size of the Nissl granules, was present in the neurons (fig. 4: 2).

(2) A 10-yr-old monkey weighing 16 kg. Sacrificed at 2 months after nerve block. The facial paralysis of the blocked side had almost completely recovered except that the nictitating reflex was slightly slower than on the normal side. In the facial nerve cells, basophil granules had reappeared next to the nuclear membrane. However, the granules were still small and the nuclei were positioned towards one side of the cells. No normal cell was found (fig. 4: 3).

(3) A 4-yr-old monkey weighing 9 kg. Sacrificed at 7 months after nerve block. No difference was visible between the blocked and unblocked sides of the face. The nerve cells in the facial nucleus were nearly normal in the blocked side, but an incomplete picture of recovery with the presence of small Nissl’s granules was observed in some cells (fig. 4: 4).

In the nerve-blocked animals, no difference in the number of nerve cells of the nucleus was evident between the blocked and unblocked sides.

DISCUSSION

Because the aetiology of idiopathic hemifacial spasm has remained unknown, no causative agent or reliable treatment has been established. Facial nerve block by alcohol injection (Harris and Wright, 1932), neuroanastomosis (Törnä, 1962) and neurotomy have been reported to be valuable procedures to control the condition. Alcohol injection and neuroanastomosis reduce hyperexcitability of the facial nerve by producing a pronounced facial palsy. The partial division of the facial nerve either at the peripheral branches (Diamant, Enfors and Wiberg, 1967) or at the major branches (Scoville, 1969) may result in severe facial palsy. The recently reported effect of facial nerve decompression at the cerebello-pontine angle requires further evaluation (Jannetta et al., 1977).

The facial nerve block method developed by Wakasugi (1972a) has been regarded as the most effective therapy for facial spasm, and many reports have supported this (Wakasugi, 1972a, b). However,
the mechanism of production of a long remission following paralysis remained unknown. This study attempted to clarify changes occurring in the nerve cells at the facial nucleus.

It is known that when the axon of a nerve cell is injured, sequential changes are produced in the body of the cell. According to Geist (1933), these changes vary depending on the following factors; (1) species, (2) age, (3) distance between the point of injury and nerve cell, (4) period of survival after operation and (5) the histological and functional type of neuron. This study was conducted using the same clinical procedure to produce a facial nerve block in mature monkeys which are considered to be anatomically close to man. The period of investigation was intended to cover the 2-month period when paralysis disappears and the 7-month period when relapse of spasm occurs. The sequence of recovery of facial paralysis after a nerve block in monkeys was about the same as that in man.

Nissl (1891) first observed a degenerative change in the nerve cells of the facial nucleus after the facial nerve was severed. A more detailed study was conducted by Cammermeyer (1955, 1963a, b, 1969). However, these studies were concerned with the consequences of cutting the facial nerve below the stylomastoid foramen and no observation was made on changes produced in the nerve by inserting a needle into the neural canal.

Torvik and Skjorten (1971) investigated the degenerative changes produced in nerve cells following crushing of the facial nerve of mice close to the stylomastoid foramen. According to their report, the nerve function of the animals recovered in 10-12 days, but it required 30-60 days for complete recovery of the central nervous system. It was also confirmed in our experiments that the nerve function recovered in 2 months but that it took 7 months for the nerve cells in the facial nerve nucleus to recover completely. On the basis of these observations, the mechanism by which a complete remission for about 6 months is produced in patients following nerve block is suggested. During this period of remission the nerve cells in the facial nucleus are in
the process of recovery and generate an optimum discharge to the peripheral nerves to produce a clinically normal state. The return of facial spasm which occurred about 8 months after the block may be a consequence of complete recovery of the nerve cells.

REFERENCES


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MODIFICATIONS DES CORPS DE CELLULES DU NOYAU FACIAL APRÈS BLOCAGE DU NERF DE LA FACE

RESUME

On a provoqué le blocage du nerf de la face sur quatre singes, en insérant une aiguille dans le tronc du nerf facial. On a étudié les modifications continues qui se sont produites dans les cellules du nerf du noyau facial: 4 jours plus tard, de même que 2 mois et 7 mois après. Bien qu'ils se soient remis de la paralysie faciale en deux mois, on pouvait toujours voir que des changements considérables s'étaient produits dans les corps de cellules du noyau facial, et dont ils ne se sont complètement remis qu'après sept mois. On considère qu'il est possible qu'une décharge optimale soit produite par ces cellules nerveuses du noyau facial pendant la période de récupération laquelle, dans la pratique clinique, pourrait entraîner une remission clinique des spasmes faciaux en six mois.

ZELLKÖRPERVERÄNDERUNGEN IM GESICHTSNUKLEUS NACH GESICHTSNERVBLOCKIERUNG

ZUSAMMENFASSUNG


CAMBIOS DE LOS CUERPOS CELULARES EN EL NUCLEO FACIAL DESPUÉS DEL BLOQUEO DEL NERVIO FACIAL

SUMARIO

Se provocaron bloqueos del nervio facial de cuatro monos mediante la inserción de una aguja en el tronco del nervio facial. Los cambios consecuentes de las células del nervio en el núcleo facial fueron objeto de investigaciones 4 días, y 2 y 7 meses después. Aunque se repusieron en 2 meses de la parálisis facial, eran evidentes aún considerables cambios de los cuerpos celulares en el núcleo facial, los que se recuperaron totalmente 7 meses después solamente. Se da por sentado que una descarga óptima es causada por esas células nerviosas en el núcleo facial en el curso del periodo de recuperación, el cual podría, en práctica clínica, resultar en una remisión clínica de 8 meses en cuanto al espasmo facial.